

# Chapter 17

## Mastitis

Timothy H. Ogilvie

### I. BOVINE MASTITIS

#### A. Overview

1. Definitions
  - a. Clinical **mastitis** is inflammation of the mammary gland characterized by changes in milk color and consistency.
  - b. Subclinical **mastitis** produces no noticeable udder inflammation or milk abnormalities but results in a high somatic cell count (SCC).
  - c. **Peracute mastitis** is a severe inflammation of the udder with a marked systemic reaction.
  - d. Acute **mastitis** is a severe inflammation of the udder with a mild to moderate systemic reaction.
  - e. Subacute **mastitis** is a mild inflammation of the udder with persistent milk abnormalities, such as changes in consistency, color, or milk production.
  - f. Chronic **mastitis** is defined as recurrent attacks of udder inflammation with little noticeable change in the milk between attacks.
2. Patient profile and history
  - a. Almost all clinical **mastitis** occurs during lactation, and 60% of cases occur during the first 6 weeks of lactation.
  - b. **Mastitis** is the most costly disease in North American animal agriculture, with an estimated 50% of dairy cows affected to some degree. However, most cases are nonclinical or subclinical, and cows vary in susceptibility.
3. Etiology and **pathogenesis**
  - a. Etiology. Most cases of **mastitis** are caused by microbial infection. Sources of infection include the udder, skin, and environment.
  - b. Pathogenesis
    - (1) In most cases, causative organisms enter the teat duct through the streak canal, multiply there, and progress upward into the **lactiferous sinus**, collecting ducts, and alveoli.
    - (2) The invading organisms cause an inflammatory response following leukocyte migration to the udder and edema.
    - (3) Resolution of the infection may result in fibrosis, abscess formation, or glandular atrophy.
4. Laboratory tests. Milk cultures should be obtained before initiating therapy so that alternate plans may be made in case of treatment failure. The culture results from a single case (e.g., *Escherichia coli*) might not reflect herd status (e.g., *Staphylococcus aureus*).
5. Therapeutic plan
  - a. A commercially available and proven **intramammary** medication for nonlactating cows should be used on all quarters of all cows at the end of lactation (drying off).
  - b. Cows with clinical signs should be treated promptly with a proven **intramammary** medication for lactating cows. Exceptions to this may be staphylococcal **mastitis** and other causes of **mastitis** nonresponsive to antibiotics (e.g., yeasts, fungi, *Mycoplasma* species, *Nocardia asteroides*). Treated cattle should be identified so that milk-withholding requirements can be **respected**.
  - c. Treatment for acute clinical **cases** should include the manual removal of as much milk as possible from infected quarters (i.e., stripping).

6. Prevention. The goal in most dairies is to maintain a herd incidence of less than or equal to 3% clinical infection, zero colony-forming units/ml on bacteriology of a bulk milk tank sample, and an SCC of 100,000–250,000 cells/mm<sup>3</sup>. An effective mastitis control program includes:

- Hygiene
  - Post-milking teat sanitation with commercially available and proven products reduces infection from contagious pathogens.
  - Premilking teat sanitation with commercially available and proven products reduces the likelihood of mastitis due to environmental pathogens (e.g., coliforms).
  - Management practices that improve overall hygiene of the environment should be employed, allowing cows to be clean, dry, and comfortable.
- Milking procedures and machine function
  - Milking machines should be installed and maintained according to the manufacturer's instructions.
  - Inflations and hoses should be changed on a regular basis.
  - Teats should be disinfected and dried with individual paper towels or washcloths before milking machine placement.
  - Teat cup sanitation should be practiced between cows.
- Accurate and up-to-date records should be maintained, and cull cows with chronic mastitis.

## B. Subacute mastitis

### 1. Streptococcal

#### a. *Streptococcus agalactiae* mastitis

##### (1) Patient profile and history

(a) History. This organism was the primary cause of mastitis in North America before antibiotic therapy, machine milking, universally accepted prevention techniques, and improved hygiene practices. It is still an important and relatively common pathogen exceeded in prevalence only by *Staphylococcus aureus*. Therefore, owners should be warned not to become complacent about the organism as a cause of mastitis because it may still result in major losses of milk.

(b) Susceptibility. The disease is often introduced into the herd by infected cows. Cows have varying susceptibilities within herds. Also, cows exhibit an increased susceptibility with age. This phenomenon, although unexplained, likely results from increased teat sphincter patency with age.

##### (2) Clinical findings

(a) Glands. Mastitis caused by *S. agalactiae* is usually nonclinical with periodic clinical flare-ups. The affected mammary gland will evidence heat, pain, and swelling. Inflammation may be mild except following the initial exposure to the organism, in which case the inflammatory reaction of the gland is moderate (subacute mastitis). If an animal is untreated, clinical cases persist with recurrent flare-ups.

(b) Milk. Although milk from affected glands may appear normal between mastitis episodes, examination of the milk during clinical periods reveals clots ("ropy" milk) in watery foremilk. Total milk yield per lactation is reduced with each recurrent bout.

##### (3) Etiology and pathogenesis

(a) Etiology. *S. agalactiae* causes a highly contagious, obligate infection of the mammary gland.

(b) Pathogenesis
 

- Transmission. This organism may survive outside of the mammary gland for a short time; therefore, it may be transmitted from cow to cow via fomites (e.g., milker's hands, multi-use towels, milking machines). The organism colonizes the teat, and infections of the mammary gland occur following passage of the organism through the teat canal.

(ii) Inflammation. The organism does not invade the glandular tissue but remains on the epithelial surface of acini and ducts, where it causes tissue damage by lactic-acid production. This epithelial inflammation results in fibrosis of interalveolar tissue and involution of the acini. Further flare-ups produce more lobular fibrosis and loss of secretory function and, thus, a decreased milk yield. Glandular atrophy is the eventual outcome.

(iii) Milk clots. As inflammation subsides during each episode, the epithelial lining of the acini and ducts sloughs. This inflammatory debris and other somatic cells result in the clinical appearance of stringy or ropey clots in the milk.

(4) Diagnostic plan

- Clinical mastitis is diagnosed by clinical finding and supported by tests that qualify and quantify udder inflammation in the individual. Bacterial isolation and identification confirm *S. agalactiae* as the causative organism.
- For a herd diagnosis, bulk tank SCC and microbiology of composite bulk milk tank samples aid in making the diagnosis.

(5) Laboratory tests

- Routine milk culture demonstrates relatively pure cultures of *S. agalactiae*. However, bacterial isolation from the milk of individual cows may be negative during the invasive phase of the initial infection or during clinical flare-ups when cell counts are high. Bulk milk samples are often used for isolation of the organism to identify or monitor herd problems.
- Indirect tests to measure increased cellularity of the milk are also used. The California mastitis test (CMT) [which measures cell nuclear protein], individual cow SCCs, and bulk tank SCCs are used to confirm and quantify udder inflammation.

(6) Therapeutic plan

- Lactating cows. Commercially available procaine penicillin G intramammary infusions are used for lactating animals (100,000 units/gland for one daily infusion for 3 days). This treatment often results in a high cure rate (90%).
- Unresponsive cows. If the cow does not respond with 3 days of therapy, consider that there might be a resistant strain of *Streptococcus*, a mixed bacterial infection, or the attainment of ineffective levels of antibiotic in the milk of a high-producing cow.
  - Resubmission of samples for culture and sensitivity is then necessary.
  - Alternative therapies include increasing intramammary penicillin dosage, changing antibiotics, or using the parenteral route. With these alternative therapies, labeled recommendations for antibiotic residues in the milk will not be valid, and milk must be discarded for more time before it is fit for human consumption.
- Dry cows. Dry cow therapy with commercial nonlactating cow products also is effective to eliminate the organism from the gland. However, this treatment usually is not necessary because of the efficacy of lactating cow therapy.

(7) Prevention. Eradication of herd infection is possible through a dedicated program of improved hygiene and intramammary infusion therapy of all cows. A mastitis control program (see I A 6) should be employed for this disease for the general production of high-quality milk. Dedication to hygiene and prevention is necessary to maintain freedom from new infections.

b. Other streptococcal causes of mastitis

- Patient profile and history. Mastitis caused by miscellaneous streptococci is of great concern as control procedures for mastitis caused by *S. agalactiae* and *S. aureus* grow more common.
- Clinical findings. These streptococci may produce acute clinical mastitis with severe udder swelling and clots in the milk. There are associated but

contaminate milk samples (individual cow or bulk tank), and this should be accounted for when interpreting culture results.

(4) Therapeutic plan. Staphylococci usually are eradicated by standard lactational or dry cow therapy.

(5) Prevention. Isolation of these organisms may indicate poor milking hygiene (e.g., udder wash, teat dipping). Therefore, a mastitis control program that focuses on hygiene and accepted udder health care procedures is recommended.

**C.** Acute and **peracute mastitis**. Many of the organisms that cause acute and peracute mastitis reside in the environment, and most are not inhabitants of the udder.

1. **Coliform mastitis** (acute mastitis, acute coliform **mastitis**, environmental **mastitis**). Coliform organisms are the most common cause of fatal mastitis and approximately 50% of cows die or are culled because of the disease. The gram-negative organisms are opportunistic and contaminate the teat between milkings.

a. Patient profile

(1) Coliform mastitis occurs most commonly within a few days of calving (0–6 weeks) and usually involves one or two quarters of the mammary gland. This disease may be seen in recumbent cattle (e.g., parturient paresis, downer cow).

(2) Susceptibility. Low levels of somatic cells in the bovine udder may increase the susceptibility of cows to coliform mastitis.

(3) Incidence. Disease usually occurs sporadically, but a high herd incidence can be 25%. There may be higher incidence in older cows, higher producing cows, or in herds that practice stringent control for subclinical mastitis. **Coliform mastitis** is most common in larger herds and herds experiencing heavy population pressures, crowding, and poor hygiene or management practices.

b. Clinical findings. Coliform mastitis causes **peracute** or acute clinical signs. Rarely does the disease progress to a prolonged course of chronic mastitis.

(1) Systemic signs may occur before changes are evident in the milk. The disorder is characterized by the sudden onset of agalactia and toxemia. Cows exhibit anorexia, severe depression, trembling, tachycardia, tachypnea, and fever or a subnormal temperature in advanced cases. Cows are weak and recumbency is common. Often, there is concurrent diarrhea and dehydration.

(2) **Affected** quarters are warm and swollen but are not gangrenous or cold as in some other **peracute mastitis** conditions. Affected glands secrete a serous fluid with small flakes that are best seen by using a strip cup.

c. Etiology and pathogenesis

(1) Etiology. *E. coli*, *Klebsiella* species, and *E. aerogenes* are discussed together and, for the purposes of this section, are collectively called **coliforms**.

(2) Pathogenesis. These organisms are transferred from the environment to the cow and produce similar (indistinguishable) clinical signs. Even though they produce acute and often fatal clinical signs, many infections by these organisms are subclinical and self-limiting.

(a) Source. Bedding and feces, usually under wet conditions, are primary sources of contamination. *Klebsiella pneumoniae* has most specifically been associated with sawdust and wood shavings bedding.

(b) Predisposing factors. Coliform bacteria on the teat end are usually transitory because the teat most often provides an effective barrier to infection. However, improper milking machine function, teat injuries (stepping on or crushing), teat sphincter relaxation with older cows, and the use of teat dilators may allow penetration by environmental pathogens. Infection also can occur in the very late dry period just before parturition. The mammary gland is most prone to infection by these organisms in early lactation and more resistant in later lactation.

(c) The systemic effects of this condition result from the elaboration of **endotoxin** from the organism or from the recruitment of host mediators, such as eicosanoids, prostaglandins, and thromboxanes. Endotoxin causes in-

creases in vascular permeability (edema) and neutrophil "pooling" in the udder. The systemic effects may lag behind the growth of the organism in the udder and parallel the massive release of endotoxin as the organisms die off.

(d) Host defense. Neutrophils may be ineffective in eliminating organisms but will limit bacterial invasion, and in the case of an adequate host response, the infection is limited to mammary sinuses with no secretory tissue involvement. The gland usually returns to normal function if the animal recovers.

d. Diagnostic plan. The diagnosis is based on the patient profile, history, clinical signs, and milk findings. Milk culture and sensitivity help define the pathogen, although it must be recognized that occasionally samples are negative depending on the effectiveness of neutrophils in clearing the organism.

e. Laboratory tests

(1) Hematology shows a severe leukopenia, neutropenia, and a degenerative left shift due to massive neutrophil pooling in the affected gland.

(2) Serum chemistry often reveals hypocalcemia.

(3) Culture results are positive for the coliform organism except in those cases where neutrophils have cleared the organism from the milk. This may occur in less acute cases.

(a) Rapid-response **culture** media are available for differentiation of gram-positive and gram-negative organisms causing clinical **mastitis** (Hy-Mast Mastitis Screening Test, Pharmacia & Upjohn Animal Health, Orangeville, Ontario, Canada).

(b) Results of milk culture may be available in 12–24 hours and allow more informal decisions to be made regarding use of intramammary antibiotic therapy.

f. Differential diagnoses include other causes of acute and **peracute mastitis**, parturient paresis (see Chapter 9), and other causes of septicemia or toxemia, such as **salmonellosis** (see Chapter 3).

g. Therapeutic plan. Acute coliform **mastitis** should be considered an emergency situation and treatment instituted accordingly.

(1) Antibiotic therapy is somewhat controversial.

(a) Studies have shown that by the time clinical signs occur, most of the organisms have been cleared from the mammary gland by the neutrophils. Also, a major die-off of bacteria in response to antibiotic use causes a further release of endotoxin, which exacerbates clinical signs.

(b) Others argue that antibiotics are necessary in cases in which neutrophils have not countered the infection or when organisms may have moved into the blood stream (bacteremia).

(c) When used, appropriate systemic antibiotics include gentamicin (2–5 mg/kg intravenously, twice daily), amoxicillin and clavulanic acid combinations, and cephalosporins.

(2) Other common treatments include:

(a) Stripping the affected quarter hourly

(b) **Oxytocin** (30 IU), which may aid in milk letdown

(c) Fluid and electrolyte therapy—balanced electrolyte solution (40–60 L during the first 24 hours) or hypertonic saline (7.5%) combined with oral fluids

(d) Anti-inflammatory agents

(i) **Corticosteroids**: 1 mg/kg intravenously twice during the first day of treatment only

(ii) **Flunixin** meglumine: 0.25–1.1 mg/kg intramuscularly or intravenously twice daily

(e) **Intramammary** antibiotics—commercially available and may be used at night after the last stripping but show little experimental **efficacy**

(f) Calcium, which should be administered carefully if necessary (perhaps subcutaneously)

- (g) Nursing care, such as rolling the cow periodically from side to side to prevent muscle necrosis, bedding the animal on a firm but cushioned surface (e.g., manure pack), and bathing and massaging the udder
- h. Prevention and control of coliform mastitis within a herd includes:
  - (1) Improving milking and premilking hygiene
  - (2) Encouraging environmental hygiene and clean bedding
  - (3) Considering other types of bedding (e.g., sand) if sawdust or shavings are implicated
  - (4) Keeping bedding dry and avoiding overcrowding
  - (5) Keeping cows standing for a time after milking
  - (6) Controlling parturient diseases (e.g., milk fever, downer cows)
  - (7) Providing calving and maternity pens to help decrease udder trauma at calving
  - (8) Administering a coliform subunit (core) bacterin against a variety of gram-negative organisms at drying off, 3–4 weeks later, and again at or near freshening

## 2. **Peracute** staphylococcal mastitis

- a. Patient profile and history. The most common presentation of staphylococcal infection of the mammary gland is as a chronic subclinical mastitis. Occasionally, infection presents as a peracute or acute mastitis seen in early lactation. In its peracute form, this infection is often fatal.
- b. Clinical findings
  - (1) There is a sudden and severe systemic reaction. The cow's temperature may be 41°C–42°C but becomes subnormal as the condition progresses. There is tachycardia, anorexia, depression, and muscular weakness resulting in recumbency, and the case may appear similar to coliform mastitis.
  - (2) The affected quarter is swollen, hard, and sore, causing lameness in ambulatory animals.
  - (3) Discoloration of the udder may be evident early (blue-black gangrene). The development of a gangrenous udder is unique to *Staphylococcus* and *Clostridium* infections. Within 24 hours, the gangrenous areas turn black and ooze serum, and subcutaneous emphysema may be evident. The milk secretion is scant with bloody, serous fluid and occasional gas. Toxemia is profound, death is rapid, and even in animals that recover, the quarter is lost. The affected quarter sloughs over time in a messy manner.
- c. Etiology and pathogenesis. *S. aureus* is the most common cause of peracute staphylococcal mastitis. The pathogenesis is as in subacute staphylococcal mastitis, but the peracute nature of the disease is likely the result of an infection in early lactation with a large inoculum of organism.
- d. Diagnostic plan. The diagnosis of peracute mastitis is made on clinical findings. The definitive diagnosis is supported by laboratory tests and culture of *S. aureus*.
- e. Laboratory tests
  - (1) Hematology shows an inflammatory leukogram.
  - (2) Clinical biochemistry indicates a severe, systemic state (shock). Fibrinogen is elevated.
  - (3) Milk culture is positive for *S. aureus*, but the information is of little relevance because of the peracute nature of the disease.
- f. Differential diagnoses include parturient paresis, coliform mastitis, *A. pyogenes* mastitis, and clostridial infection of the udder.
- g. Therapeutic plan. Generally, treatment is very expensive, and cows that recover are often culled because only three viable quarters remain. The life of the animal may be saved with early and aggressive therapy. The following treatments can be considered:
  - (1) Intravenous crystalline penicillin or tetracycline followed by subsequent intramuscular treatment
  - (2) Intravenous fluids
  - (3) Frequent massage, bathing, and milk stripping from the affected quarter  
(Note: oxytocin has little effect on milk letdown in this instance.)

- (4) Amputation of the quarter, which is usually a final and heroic attempt to save the cow's life
- (5) Intramammary antibiotic infusions have little effect because of the edema, swelling, and gangrenous necrosis of the udder.
- h. Prevention. Little can be done to prevent this condition other than maintenance of udder health as recommended in I B 2 a (7).
- 3. Acute streptococcal mastitis
  - a. Etiology and clinical findings. Infection with *S. dysgalactiae*, *S. uberis*, *S. viridans*, *S. pyogenes*, *S. pneumoniae*, and other streptococci nonagalactiae organisms may produce an acute mastitis with swelling of the individual quarter. There may be a moderate systemic reaction, and milk secretion is abnormal with evidence of flakes and clots. Specifically, *S. pneumoniae* will produce a peracute mastitis.
  - b. Therapeutic plan. The disease should be treated like other cases of acute mastitis (see I C 1 g).
- 4. *Actinomyces* pyogenes mastitis
  - a. Patient profile and history
    - (1) *A. pyogenes* mastitis can occur in any cow but is distinguished from other types of mastitis because it usually occurs in dry cows or pregnant heifers. Mortality is high in cattle, and glandular function is lost.
    - (2) This infection is seen most commonly in Europe during summer months (hence the name summer mastitis) and in extensive dairy production areas (e.g., New Zealand, Australia). The disease is seen in North America if seasonal calving is practiced in dairy operations.
    - (3) This sporadic form of mastitis occurs most often in pastured cattle under conditions of wet summers, high fly populations, and poorly observed animals.
  - b. Clinical findings
    - (1) This peracute mastitis causes severe, systemic reactions. The cow's fever reaches 40°C–41°C. There is tachycardia, anorexia, depression, and weakness. Abortions may occur.
    - (2) The udder is hard, swollen, and painful. An initial watery secretion becomes purulent and foul smelling. This secretion is subjectively diagnostic for the disease.
    - (3) In cows that survive the initial systemic reaction, the quarter becomes very hard, and abscesses develop, which rupture at the supramammary lymph nodes or at the base of the teat. Gangrene does not occur, but the quarter sloughs.
  - c. Etiology and pathogenesis. *A. pyogenes* may act alone or with other organisms to produce a suppurative mastitis. The organism is thought to be spread by flies and introduced via the teat canal.
  - d. Diagnostic plan and laboratory tests. Clinically, the disease is distinctive, and the causative organism can be recovered on milk culture.
  - e. Therapeutic plan. Therapy has little effect in clinical cases. If considered, treatment should include:
    - (1) The systemic use of broad-spectrum antibiotics
    - (2) Stripping the affected quarter every 1–2 hours in the initial phase of the disease
    - (3) Supportive care (e.g., fluid therapy, rolling, deep bedding)
  - f. Prevention. In places where the disease is prevalent (e.g., New Zealand, Australia), dry cow treatment may need to be performed 2–3 times during the dry period. There must be strict attention to fly control and closer observation of cows on pasture.
- 5. *Nocardia* mastitis
  - a. Patient profile. This is a relatively uncommon type of mastitis but has been isolated periodically in herds. This disease is usually sporadic and most common in early lactation.
  - b. Clinical findings
    - (1) With infection early in lactation, there is severe systemic involvement often

resulting in death of the cow. If not fatal, the infection may resolve to chronic mastitis and udder fibrosis with draining, suppurative sinus tracts.

- (2) Infection **later** in lactation may produce acute inflammation with only moderate systemic involvement. There is still glandular fibrosis with the presence of discrete nodules discernable on palpation of the gland. The milk appears greyish with clots, white granules, or both.
- (3) Subclinical infections may occur but do not differ significantly from other types of subclinical mastitis.

c. **Etiology and pathogenesis.** *Nocardia asteroides*, the causative organism, is a common soil contaminant. If disease is a herd problem, there is likely a common method of introduction (e.g., multi-injection mastitis preparations, cracked milking machine liners). When the gland is invaded by the organism, extensive glandular destruction and fibrosis occur. Local invasion of other tissues may occur (lymph nodes). There is eventual loss of production, and the cow is **inevitably culled** if she survives.

d. **Diagnostic plan.** Clinical findings are highly suggestive of the condition, and the causative organism is confirmed by microbiology.

e. **Therapeutic plan.** Therapy is unrewarding. Long-term treatment (1–2 weeks) of erythromycin and miconazole would be **necessary** but is of limited value. The organism is specifically resistant to neomycin-based intramammary products.

f. **Prevention.** Milking and environmental hygiene is all that can be recommended for the herd. Culture-positive cows should be culled.

#### 6. *Mycoplasma mastitis*

##### a. Patient profile and history

- (1) Cows at any age or stage of lactation may be affected, but those in early lactation show the most severe systemic signs. The disease occurs worldwide and is most common in large, intensively managed herds with constant movement of cattle onto the premises.
- (2) Historically, incidence of disease was thought to be associated with the use of multi-dose vials of intramammary products. Now, disease is thought to occur in association with shedder cows or outbreaks of pneumonia, urogenital disease, and **polyarthritis** in heifers and calves.

##### b. Clinical **findings**

- (1) In lactating cows, a sudden drop in milk **production** and a painless udder swelling occur (edema). Supramammary lymph nodes may enlarge. All four quarters may be affected.
  - (a) **Systemic findings.** A mild fever and anorexia is seen with cows in early lactation; otherwise, there may be little systemic involvement.
  - (b) The udder secretion appears normal in the early course of disease, but if left to stand, a fine, grainy or flaky precipitate settles to the bottom of the sample container. As the disease progresses, the milk assumes a cheesy consistency in chronic cases.
  - (c) **Duration.** Cases may persist for weeks. Depending on the **type** of *Mycoplasma* involved, the gland may atrophy for the current and subsequent lactations or return to normal function.
- (2) Dry cows show little udder swelling.
- (3) **Other** cows or young dairy animals in the herd may show arthritis or **pneumonia**.

##### c. Etiology and pathogenesis

- (1) **Etiology.** Many species of *Mycoplasma* may produce mastitis, but ***Mycoplasma bovis*** is the most common causative agent.
- (2) **Pathogenesis**
  - (a) **Source.** *M. bovis* produces a purulent interstitial mastitis. It may be introduced to a herd or survive in shedder cows that have **recovered** from disease. The source of **shedding** may be from the udder, vagina, or upper respiratory tract.
  - (b) **Route of infection.** *M. bovis* may reside on mucous membranes and gain entrance to the gland via the teat canal or systemically through aerosol.

spreading hematogenously. If the organism invades the udder via the teat, the infection may spread to other quarters and sites hematogenously.

- d. **Diagnostic plan.** The specific clinical findings related to the udder, other clinical findings in the herd (e.g., pneumonia, arthritis, vaginitis), and laboratory results collectively form the diagnosis.
- e. **Laboratory tests.** *Mycoplasma* species can be cultured from the milk of affected quarters, but this test is not routinely performed by many laboratories. In individually affected animals, there is a marked leukopenia and a very high SCC (more than 20 million/ml).
- f. **Therapeutic plan**
  - (1) **Systemic antibiotics.** Therapy is unrewarding, but attempts to cure the condition may be made with systemic antibiotics (e.g., tetracycline, erythromycin, tylosin). Residue avoidance and labeled restrictions for use in dairy animals must be considered.
  - (2) **Response to therapy.** It is difficult to advise on the eventual course of the disease because outcomes are variable. While individual animals may recover health and function, many other cows fail to recover. Also, herd outbreaks occur, which necessitates culling many animals.
- g. **Prevention** includes closing the herd, establishing a routine udder health and **mastitis** control program, and monitoring incidence of infection via bulk milk tank culture. Some practitioners advocate the use of an autogenous vaccine in chronically affected herds.

#### D. Other causes of mastitis

1. ***Mycobacterium bovis.*** Mastitis resulting from *M. bovis* infection is a public health hazard because contaminated milk may be macroscopically normal but can act as a source of human tuberculosis. Fortunately, many jurisdictions are free of this disease because of active regulatory efforts (i.e., identification and slaughter) carried out in the past. *M. bovis* (the causative agent for tuberculosis in cattle) causes a hard, swollen udder and, on occasion, enlarged, supramammary lymph nodes. This disease occurs with other systemic findings (e.g., pneumonia, chronic ill-thrift).

2. **Fungi and yeasts.** Yeasts are more common than fungi, and ***Candida*** species are the most often implicated. Infection may persist for weeks and may resolve or produce udder damage, resulting in **cullage** of the affected individual.

- a. **Clinical findings**
  - (1) There is little systemic reaction with this mastitis, although there may be some fever and anorexia.
  - (2) The udder inflammation is acute, with marked swelling of the affected quarters and enlargement of the supramammary lymph nodes.
  - (3) **Milk.** There is a significant drop in milk production, and the milk appears viscous, white-grey, and mucoid in consistency.

- b. **Etiology and pathogenesis.** ***Cryptococcus neoformans*** and ***Aspergillus fumigatus*** are other reported yeasts causing mastitis. The use of contaminated infusion material or overzealous use of intramammary antibiotic therapy may result in herd outbreaks. Also, there is a growing body of subjective evidence that implicates yeast contamination of the udder from growth in bedding during periods of moisture and warmth.

- c. **Therapeutic plan.** The best advice is to discontinue all antibiotic therapy and strip out the quarter four times daily.
  - (1) **Yeast mastitis** is not responsive to therapy, but the infection is self-limiting in some cows in 30–60 days, particularly those animals infected with *Candida* species.
  - (2) The use of intramammary antibiotics is contraindicated in this condition.

3. **Algae**

- a. **Clinical findings and etiology.** ***Prototheca trispora*** and ***Prototheca zopfii*** have been identified as causing chronic, clinical mastitis in cattle. The organisms are isolates from the environment and produce glandular swelling, decreased milk

production, and large clots in watery milk. There is a progressive abscessation of the gland, and the cow must be culled.

b. Therapeutic plan. There is no successful treatment.

**4. *Pseudomonas aeruginosa***

a. Clinical **findings** and **pathogenesis**. This organism may produce either an acute mastitis with a systemic reaction or a chronic mastitis. Glandular function may be lost. Usual sources of infection include contaminated infusions or environmental sources (e.g., contaminated udder wash water).

b. Therapeutic plan. Third-generation cephalosporins are recommended for treatment, but success may be limited.

**5. Bacilli**

a. Clinical **findings** and **etiology**. *Bacillus cereus* and *Bacillus subtilis* may cause an acute hemorrhagic mastitis. Teat and udder trauma are precipitating events for the introduction of the organism spores. Proliferation of the organisms produces a peracute, toxic mastitis. Gangrene follows, with eventual loss of the quarter if the cow lives.

b. Therapeutic plan. Treatment must be intensive and heroic, consisting of intravenous fluids, broad-spectrum antibiotics, anti-inflammatory agents, udder massage, and frequent quarter stripping. Results are often disappointing.

**6. *Clostridium perfringens***

a. Clinical **findings** and **etiology**. *C. perfringens* type A may produce a peracute, highly fatal toxic mastitis, which is characterized by gangrene and a thin, brown glandular secretion accompanied by gas.

b. Therapeutic plan. Early treatment with intravenous crystalline penicillin may be successful.

**II.**

**EQUINE MASTITIS.** Mastitis in mares is rare. What is more common is swelling and soreness of the udder due to failure of the foal to nurse. A lower incidence of mastitis in mares as compared with dairy cows may be explained by the smaller size of the udder and teats. In addition, there is less exposure to physical trauma, milking machines, and contaminated fomites, and the gland is emptied relatively often because of the frequent nursing of the foal.

**A.** Clinical finding The udder is **enlarged**, warm, firm, and painful on palpation. Both halves of the udder are often affected simultaneously. The milk is abnormal (thin, discolored and with clots). Glandular edema may extend forward along the ventral abdomen.

**B.** **Etiology.** When mastitis does occur (e.g., at foal weaning), culture may reveal a **causative** organism. *S. zooepidemicus* is the most common isolate, but many other **pathogens**, including gram-negative organisms, have been isolated in cases of mare mastitis.

**C.** Differential **diagnoses** should include mammary gland neoplasia.

**D.** Therapeutic plan. Response to therapy is usually **good**. Therapeutic measures include systemic antibiotics (if the mare is ill), warm udder compresses, frequent stripping using oxytocin, and hydrotherapy.

1. The choice of antibiotic therapy should be made on the basis of milk culture and sensitivity. In the absence of this information, broad-spectrum antibiotics should be used.
2. Bovine **intramammary** antibiotic infusions may be used in cases of mild to subacute infections.



**OVINE AND CAPRINE MASTITIS.** Subacute and chronic forms of mastitis are most common, but acute types of the disease have the most clinical importance.

**A. *S. aureus* mastitis.** *S. aureus* is the most common cause of mastitis in sheep and goats.

1. Patient profile and history. This disease is most common in animals on pasture and is identical with the bovine peracute form.
2. Clinical **findings** include systemic signs: watery, bloody-brown secretion, udder edema, and gangrene.
3. **Pathogenesis.** The organism usually gains access via teat injuries from suckling lambs or kids. The source of infection is contaminated pastures or bedding.
4. Therapeutic plan. Treatment is **unrewarding**, and the mortality rate is high in affected animals. Methods of treatment are identical to those used for cows with peracute mastitis (i.e., fluids, broad-spectrum antibiotics, frequent stripping).
5. Prevention consists of culling affected ewes and pasture rotation. Also, in areas where small ruminant dairying is of economic importance, vaccination with a staphylococcal bacterin and drying-off therapy with intramammary antibiotics may be of value.

**B. *Mycoplasma* mastitis** is known as contagious **agalactia**. This disease is seen in southern Europe and northern Africa. Causative agents include *M. agalactiae* and *M. mycooides* var *mycooides* in goats. Infection causes mastitis, arthritis, ophthalmitis, and abortion. The mortality rate is high (10%–30%), and the udder is permanently **damaged**.

**C. *Pasteurella hemolytica* mastitis**

1. Patient profile and history. Mastitis caused by *P. hemolytica* is common in ewes, although it is sporadic in occurrence. The disease is peracute, **gangrenous**, and likely results from teat injuries from **suckling** lambs.
2. Clinical finding Glandular swelling and toxemia are **cardinal signs**. Hind limb lameness may be an early and **diagnostic sign**. The udder is initially hot, swollen, and painful but rapidly becomes cold, blue, and **gangrenous**. Death occurs due to toxemia. If the animal survives, the udder will slough. Pneumonia may occur concurrently in the lambs.
3. **Pathogenesis.** Transmission is from infected soil or **bedding** but does not seem to be related to poor **hygiene**. There is evidence that some ewes may act as chronic carriers for *P. hemolytica*.
4. **Therapeutic** plan. Broad-spectrum antibiotics may be an effective treatment if administered early.
5. Prevention. Control procedures include **culling** recovered ewes, **autogenous** vaccination, and pasture rotation.

**D. *S. agalactiae* mastitis** in sheep and goats is similar to that in cows (see I B 1 a).

**E. Caprine arthritis encephalitis mastitis.** Udder changes are part of the viral disease syndrome known as caprine arthritis encephalitis (CAE) [see Chapter 13 III D]. CAE is associated with a hard udder developing soon after kidding. No milk can be expressed. Recovery is incomplete with this chronic disease syndrome.



**IV. PORCINE MASTITIS**

**A. Patient profile**

1. Mastitis occurs in sows at **farrowing** and is most common in mature sows that are

raised in intensive husbandry situations (e.g., farrowing crates). The condition is sporadic, and although mortality in sows is low, piglet losses are high because of starvation and crushing.

- 2 Risk factors include increasing age, overnutrition, confinement housing, poor sanitation, and large litters.

#### B. Clinical findings

1. Appearance
  - a The sow may appear normal immediately **post** farrowing, but she soon loses interest in the piglets and will lie in sternal recumbency rather than adopting a nursing posture. The sow may appear nervous and agitated.
  - b. The piglets are restless and squealing, and they wander about the pen and drink surface water. If allowed by the sow to suckle, piglets will nuzzle the udder but are unsuccessful in nursing.
- 2 Systemic findings. Sows are anorexic and feverish (39.5°C–41°C). The condition is commonly associated with constipation. Despite the historical name for this condition (mastitis-metritis-agalactiae or MMA), metritis is not a feature. The mucoid, white vaginal discharge, which is present in most sows **post** farrowing, is a normal occurrence. The glands are mottled red and yield very little milk on manual expression.

3. **Local findings.** Most of the mammary glands are swollen, painful, and hot.

#### C. Etiology and pathogenesis

1. Etiology. The majority of cases are caused by **coliform organisms** (e.g., *E. coli*, *K. pneumoniae*).
2. Predisposing factors. The likelihood of disease may include thyroid dysfunction, failure of prolactin release, or eclampsia.
3. Pathogenesis. Endotoxin release from the dying organisms in the udder is postulated to produce the clinical signs similar to acute coliform mastitis in dairy cattle (see I C 1).

D. Diagnostic plan. The condition is unique and **diagnosed** on clinical findings. **Laboratory** diagnostics are not commonly used in practice.

E. Differential diagnosis. First-litter gilts may present with glandular **changes** similar to those of mastitis, but the swelling is associated with **agalactiae** only, and the gilt is not **systemically ill**.

#### F. Therapeutic plan

1. Sow. Treatment consists of systemic antibiotics, oxytocin, and **corticosteroids**. Antibiotics should be chosen for their broad-spectrum activity, but in practice, even penicillin (20,000 IU/kg intramuscularly twice daily) has proved effective when combined with oxytocin (20–40 IU intramuscularly) and **dexamethasone** (20 mg intramuscularly) for 3 days.
2. Piglets may need supplemental nutrition, such as milk or electrolytes plus glucose, if treatment is delayed, but response of the sow is often rapid enough to allow the piglets to be successful at nursing if treatment is instituted early.

G. **Prevention** should center on cleanliness of the **farrowing** environment and of the sow before farrowing.

1. Crates should be disinfected between **farrowings** and the sow washed with soap and water as she is moved into the farrowing crate. Allow the sow ample time (1 week) to become used to her environment before farrowing, and do not institute major feeding changes.
2. **Prophylactic** antibiotics may be necessary and effective in herds with ongoing problems.

## STUDY QUESTIONS

DIRECTIONS: Each of the numbered items or incomplete statements in this section is followed by answers or by completions of the statement. Select the ONE numbered answer or completion that is **BEST** in each case.

1. The majority of clinical mastitis cases in dairy cattle in North America are:
  - acute or peracute in clinical expression.
  - seen in the first 6 weeks of lactation.
  - seen in heifers.
  - caused by trauma.
  - associated with low somatic cell counts (SCCs).
2. An accepted program of mastitis control and prevention includes:
  - dry cow therapy for all cows at the end of lactation.
  - parenteral antibiotics for all fresh cows.
  - conversion to "automatic take-off" milking machines.
  - rinsing the udder immediately before teat-cup placement.
  - vaccination of cows with a *Escherichia coli* subunit bacterin at freshening.
3. Which one of the following terms fits the definition of a **mastitis** that causes a severe inflammation of the udder with a marked systemic reaction?
  - Acute mastitis
  - Chronic mastitis
  - Subacute mastitis
  - Peracute mastitis
  - Subclinical mastitis
4. Which one of the following statements regarding *Streptococcus agalactiae* is true?
  - It is an environmental pathogen that causes mastitis.
  - It most often causes acute clinical mastitis.
  - It does not invade glandular tissue.
  - When established in a herd, it requires dry cow therapy for elimination.
  - It has been eradicated from most dairy herds in North America.
5. Which one of the following statements regarding **coliform mastitis** is true?
  - It is a contagious **mastitis** spread from cow to cow.
  - It results from exotoxins elaborated by **coliform** bacteria.
  - It produces signs of toxemia and gangrene of the mammary gland.
  - It produces a thick, purulent udder secretion.
  - It may resolve with little damage to the udder.
6. In animals with **peracute** staphylococcal mastitis, the **affected** mammary gland:
  - is hard, painful, and discolored.
  - secretes caseous, purulent material.
  - rapidly infects the other three quarters.
  - returns to full function after recovery.
  - responds to stripping aided by oxytocin.
7. Which one of the following statements regarding **peracute** staphylococcal **mastitis** is true?
  - It is best diagnosed by milk culture.
  - It is the most common presentation of intramammary infection with *Staphylococcus aureus*.
  - It is a disease of dry cows.
  - It is best treated with intravenous antibiotics and electrolytes.
  - It is usually spread from cow to cow through pulmonary secretions.
8. Which one of the following statements regarding **summer mastitis** is true?
  - It is a disease of housed dairy cattle.
  - It is most commonly transmitted by milking machines.
  - It is a disease of dry cows caused by *Actinomyces pyogenes*.
  - It is a subclinical **mastitis** of goats.
  - It is seen most often in young mares.

9. Yeasts and algae are opportunistic pathogens that may cause mastitis. How are these pathogens similar?

- (1) Both produce a severe systemic reaction.
- (2) Neither has any effect on milk production.
- (3) Both produce ropey clots in watery milk.
- (4) Neither causes any external glandular reaction.
- (5) Neither is responsive to antibiotic therapy.

10. Which one of the following statements regarding equine mastitis is true?

- (1) It should be treated with frequent stripping of the affected glands.
- (2) It is a common disease.
- (3) It is caused most commonly by *Streptococcus equi*.
- (4) It causes little observable udder change.
- (5) It most commonly causes a thick, purulent glandular discharge.

11. Which one of the following statements regarding *Pasteurella hemolytica* mastitis is true?

- (1) It is the most common cause of mastitis in sheep and goats.
- (2) It is transmitted from lambs to ewes through suckling and teat injuries.
- (3) It is known as contagious agalactiae.
- (4) It produces a chronic, subacute mastitis.
- (5) It is a federally reportable disease.

12. Which one of the following statements regarding mastitis in sows is true? Porcine mastitis

- (1) is of little clinical importance.
- (2) occurs most commonly at weaning.
- (3) is treated with intramammary antibiotics.
- (4) is similar in many respects to coliform mastitis of cattle.
- (5) occurs concurrently with metritis.

## ANSWERS AND EXPLANATIONS

1. The answer is 2 [I A 2 a]. Most cases of clinical mastitis are seen in multiparous cows early in lactation. Subclinical mastitis is of greatest prevalence, but of the clinical presentations, the order of occurrence (greatest to least) is subacute, acute, peracute. Mastitis is almost invariably caused by pathogenic bacteria and is associated with high cell counts in the milk of affected cows.

2. The answer is 1 [I A 4, 5]. Blanket dry cow therapy is recommended to treat pathogens causing clinical and subclinical mastitis (e.g., *Staphylococcus aureus*). Also, dry cow therapy reduces the risk of introducing new infections during a susceptible period of the mammary gland. Treatment of fresh cows with antibiotics is not recommended for control of mastitis. Automatic take-off milking machines afford no better control of mastitis than use of any regularly maintained, properly functioning milking system. Cows' udders should be dry before placement of teat cups to limit bacterial spread from contaminated teat ends. There are some vaccines that have proven efficacious in the prevention and control of mastitis, but an *Escherichia coli* subunit bacterin should be administered in three doses (two at and during the dry period) to allow adequate immunity to develop.

3. The answer is 4 [I A 1]. Peracute mastitis is a mastitis that causes a severe inflammation of the udder with a marked systemic reaction.

4. The answer is 3 [I B 1 a (3) (b) (ii)]. *Streptococcus agalactiae* does not invade glandular tissue, but through the effects of the lactic acid that it produces, it does produce epithelial destruction, resulting in lobular fibrosis and glandular atrophy. It is an obligate, contagious pathogen of the bovine udder and is most often expressed clinically as a subacute mastitis. It is still a relatively common mastitis pathogen (second only to *Staphylococcus aureus*) but can be eradicated through lactational therapy and strict hygienic and control measures.

5. The answer is 5 [I C 1 c]. This environmental mastitis results from contamination of the teat end by feces and contaminated bedding. The teat has often suffered damage that al-

lows entry of the organism. The udder becomes warm and swollen, but not gangrenous, and secretes a thin, watery fluid. There is little quarter damage, and most of the systemic disease is caused by endotoxins (cell-wall lipopolysaccharides) released as bacteria die.

6. The answer is 1 [I C 2 b (3)]. The gland is hard, painful, swollen, discolored, and gangrenous. It secretes a thin, brown fluid often accompanied by gas. A single gland is usually affected, and infection remains limited to that gland. Because of the necrotizing effects of the *Staphylococcus* toxins, the affected gland does not return to function even if the cow survives. Stripping is a recommended therapy, but the cow does not respond to oxytocin because of the morphologic changes induced by the toxins on the glandular tissue of the udder.

7. The answer is 4 [I C 2 g]. The peracute nature of the disease demands a diagnosis be made based on clinical signs so that treatment can be instituted promptly. It requires vigorous treatment with intravenous fluids (balanced electrolytes) and antibiotics. It is a sporadic disease of lactating cows, with the most common manifestations of *S. aureus* mastitis appearing as subclinical and then subacute mastitis. The organism is transmitted via fomites (e.g., milking machines), with the source often being mastitic glands of other cows in the herd.

8. The answer is 3 [I C 4 c]. *Actinomyces pyogenes* mastitis (summer mastitis) is a disease of dry cows in herds raised extensively (i.e., on pastures with limited observation). The organism is transmitted by flies, so fly control and hygiene are important methods of prevention.

9. The answer is 5 [I D 2 3]. Neither organism is responsive to antibiotic therapy; in fact, antibiotics are contraindicated. There is little systemic reaction with either disease. Although algae usually produce large milk clots in watery milk, yeasts produce viscous, mucoid secretion. Both organisms cause a significant glandular reaction, and both cause a significant drop in milk production.

10. The answer is 1 [II D]. Mastitis is a relatively infrequent occurrence in mares. When it does occur, it produces glandular swelling and a thin, discolored discharge. Frequent stripping and systemic or intramammary therapy is recommended. *Streptococcus equi* causes "strangles," whereas *S. zooepidemicus* is a common isolate in cases of mare mastitis.

11. The answer is 2 [III C]. *Pasteurella hemolytica* is most often associated with pneumonia but may act as a cause of mastitis when transmitted from lambs to ewes through teat injuries from suckling. It is less common as a cause of mastitis than *Staphylococcus aureus* but produces an acute gangrenous mastitis.

*Mycoplasma* organisms produce the mastitis known as contagious agalactiae.

12. The answer is 4 [IV C 2]. The causative agents are most commonly coliforms, which produce signs of endotoxemia similar to that in dairy cattle. Mastitis is a clinically important disease in terms of the health of the sow and her piglets, who do not receive adequate nutrition during the mastitis episode. This disease most frequently occurs during early lactation and requires systemic therapy with antibiotics, corticosteroids, and oxytocin. Although metritis was once thought to be part of the mastitis complex, it is no longer considered to be involved.